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LETTER TO THE EDITOR

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SARS-CoV-2–associated Guillain-Barré syndrome with dysautonomia

Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory coronavirus 2 (SARS-CoV-2), is associated with Guillain-Barré syndrome (GBS).[1–8] We describe a patient with quadriplegic GBS with dysautonomia preceded by mild COVID-19–induced diarrhea.

A 72-year-old man with coronary artery disease, hypertension, and alcohol abuse presented to a tertiary hospital with symmetric paresthesias and ascending appendicular weakness. Seven days earlier he had mild diarrhea, anorexia, and chills, without fever or respiratory symptoms. This condition resolved in 5 days. Weakness began 6 days after diarrhea, and the patient presented 1 day after neurological symptom onset.

On admission, he was afebrile with normal vital signs. Mental status and cranial nerves were normal. Strength was 4/5 for neck flexion, and 3/5 for proximal upper and lower extremities bilaterally. Tendon reflexes were absent. Sensation to light touch was diminished to wrists and knees bilaterally.

Laboratory studies demonstrated a white blood cell (WBC) count of 12 000 cells/μL (granulocytes 84%), sodium 143 mmol/L, potassium 3.1 mmol/L, blood urea nitrogen (BUN) 16 mg/dL, creatinine 0.77 mg/dL, alanine transaminase 38 IU/L, aspartate transaminase 29 IU/L, alkaline phosphatase 76 IU/L, albumin 3.7 g/dL, and total bilirubin 0.8 mg/dL. Serum thiamine, vitamin B_{12}, hemoglobin A1C, thyroid panel, creatine kinase, Lyme screen, and serum and urine protein electrophoreses were normal. Anti-ganglioside GM1, GD1b, and GQ1b and acetylcholine receptor binding, voltage-gated calcium channel, antinuclear, and antineutrophil cytoplasmic antibody titers were negative. Nasopharyngeal SARS-CoV-2 polymerase chain reaction (PCR) was positive. Chest X-ray showed mild bibasilar atelectasis vs patchy consolidations. Computed tomography of the head was normal. Incompatible implant precluded magnetic resonance imaging.

On day 3, the patient developed respiratory distress with a negative inspiratory force of −20 cmH_2O and vital capacity of 1350 mL. He was transferred to the intensive care unit and intubated. He remained afebrile and followed commands. Oxygen saturation was normal on ventilator settings positive end-expiratory pressure 5 cm H_2O and fraction of inspired oxygen 30%. Chest X-ray was stable. He was diagnosed with GBS and received intravenous immunoglobulin 2 g/kg between days 3 and 6. On day 4, he developed dysautonomia with hypotension alternating with hypertension and tachycardia. On day 6, his strength decreased to 1/5 for neck flexion, 1/5 for distal upper extremities bilaterally, and 0/5 for proximal upper extremities and throughout the lower extremities bilaterally. On day 8, he developed the syndrome of inappropriate antidiuretic hormone secretion (SIADH), with serum sodium 134 mmol/L and osmolality 271 mOsm/kg correlated with urine sodium 43 mmol/L and osmolality 731 mOsm/kg. He had no prior history of SIADH.

Cerebrospinal fluid (CSF) collected on day 8 showed WBC 1 cell/μL and protein 313 mg/dL; CSF SARS-CoV-2 PCR, Gram stain and culture, herpes simplex, varicella zoster, and cytomegalovirus viral PCRs; immunoglubulin G index; oligoclonal bands; and cytology were negative. On day 10, his oropharyngeal secretions increased, and chest X-ray showed new right lower lobe consolidation. Sputum culture grew *Stenotrophomonas maltophilia*, an organism associated with ventilator-associated pneumonia in immunocompetent hosts. WBC increased to 20 400 cells/μL (granulocytes 88%), with improvement to 9400 cells/μL (granulocytes 69%) after 2 days on sulfamethoxazole-trimethoprim. On day 12, strength improved to 3/5 for neck flexion and 2/5 throughout all limbs bilaterally. Nerve conduction studies on day 13 showed diffusely decreased velocities, conduction block, and absent F waves, supporting demyelinating GBS (Table 1). Resting needle electromyography was normal, but there was poor effort. On day 19, tracheostomy and percutaneous endoscopic gastrostomy tubes were placed. He did not develop signs of active COVID-19 infection and repeat nasopharyngeal SARS-CoV-2 PCR was negative on day 28. He remains in the ICU with severe weakness.

We have described a patient with SARS-CoV-2–associated quadriplegic GBS who had preceding diarrhea and was otherwise asymptomatic on presentation. Other patients with SARS-CoV-2–associated GBS presented with symptoms of upper respiratory infection, and some had a pre-existing diagnosis of COVID-19.[2–8] Characterizing SARS-CoV-2 clinical phenotypes has been hindered by limited testing, and the prevalence of asymptomatic or mildly symptomatic carriers is likely underestimated. This case illustrates the importance of SARS-CoV-2 testing in patients with GBS.

Dysautonomia and SIADH were prominent features in this patient. To date, these have been uncommon in other patients with SARS-CoV-2–associated GBS. An analysis of COVID-19 patients in Wuhan, China, showed that those with central nervous system symptoms had lower lymphocyte and platelet counts and higher BUN levels compared with those with peripheral nervous system or no neurological symptoms.[9] Although these laboratory values in the patient we described are similar to those seen in the Wuhan cohort with peripheral nervous system or no neurologic complications, the underlying pathophysiology predisposing certain COVID-19 patients to

TABLE 1 Nerve conduction study results

		Amplitude		Latency (ms)		Velocity (m/s)		F wave	
Nerve	Stimulation site	Right	Left	Right	Left	Right	Left	Right	Left
Ulnar (s)	Wrist	NR	–	NR	–	NR	–	–	–
Sural (s)	Calf	NR	NR	NR	NR	NR	NR	–	–
Ulnar (m)	Wrist	1.2	–	4.3	–			–	–
	Below elbow	0.6	–	9.4	–	37	–		
	Above elbow	0.4	–	14.3	–	18	–		
Peroneal (m)	Ankle	0.8	1.8	10.2	9.3			NR	–
	Below fibular head	0.4	0.3	23.4	21.3	23	24		
	Popliteal fossa	0.4	0.2	27.7	25.6	17	19		
Tibial (m)	Ankle	3.5	2.5	8.9	9.9			–	NR
	Popliteal fossa	1.0	0.3	24.6	23.0	24	28		

Note: Amplitudes are reported in millivolts for motor studies.
Abbreviations: m, motor study; NR, not reported; s, sensory study.

neurological complications remains unclear. Continued investigation of neurological sequelae is needed.

KEYWORDS

AIDP, autonomic dysfunction, COVID-19, Guillain-Barré syndrome, SARS-CoV-2

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ETHICAL PUBLICATION STATEMENT

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines. None of the authors has any conflict of interest to disclose.

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